ure of several medical attempts to relieve the dystocia, ovariosalpingectomy was performed and recovery was uneventful.

There are many possible explanations for the failure of propranolol and prostaglandin $F_{2\alpha}$ to induce ovipositon in this case. Since the propranolol dose used in this case was much lower than that used by Gross, *et al*,[1] insufficient dosage is a definite possibility. For example, if the propranolol dose in this case was allometrically scaled from the effective *Sceloporus* dose, rather than the intravenous canine dose, the iguana would have received 0.25 mg/kg (33 times more than given in this case). In addition, the previously administered oxytocin could have lowered oviductal sensitivity to the subsequent medications.

Although the serum calcium level was considered to be normal and supplemental calcium was given, hypocalcemia may have been a factor in the failure. It has been observed that reproductively active female reptiles may have serum calcium levels two to four times greater than "normally" reported for the species.[8] Whether these elevated calcium levels are essential for normal oviposition is unknown.

While the nest chamber provided for the iguana was based on a reportedly successful design, Barten[9] has pointed out the difficulty in stimulating the complex nesting system used by wild iguanas. Unsuitability of the nest environment may have overridden the effects of the treatment. Finally, other factors such as species variation in drug sensitivity, or preexisting oviductal pathology may have contributed to the failure.

The cause of the open-mouth breathing noted in this case is unclear. It is possible, however, that it was an adverse effect of either of the medications, similar to those discussed above. The author has also observed such behavior in iguanas as a threat display or as a result of hyperthemia.

Further research on the use of sympatholytic drugs for stimulation of oviposition in reptiles is warranted. With the general inavailability of arginine vasotocin and the only partial efficacy of oxytocin, another medical option would be clinically useful. Of particular interest is the possible synergistic effect of sympatholytics, prostaglandins and neurohypophyseal hormones. For example, could we increase the reliability and efficacy of oxytocin by pretreating with propranolol? Effective doses of propranolol and its cardiovascular effects in reptiles should be established. Other clinicians who have tried these alternative protocols are encouraged to communicate their findings.

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Ivermectin for the Treatment of Pentastomids in a Tokay Gecko, *Gekko gecko*.

A recently imported adult female Tokay gecko, *Gekko gecko*, was evaluated for the persistence of pentastomid ova in the feces. The lizard ate well and had no abnormal findings on the physical examination. The lizard's body weight was considered normal. Fenbendazole (Panacur® suspension 10%, Hoescht-Roussel Agri-Vet Co., Somerville, NJ), praziquantal (Droncit® 56.8 mg/ml, Miles Inc., Shawnee, KS), and ivermectin (Ivomec® 1% injection, Merck Ag Vet, Rahway, NJ), were given orally at 100 mg/kg, 10 mg/kg and 200 µg/kg respectively every two weeks for three treatments. This protocol

was successful in eliminating an oxyurid ova but not the pentastomid ova. Daily fecal flotation exams showed the continued presence of the pentastomid ova. The ivermectin dose was increased to 400 µg/kg orally every two weeks for three treatments. This dose was also unsuccessful. Several weeks later the lizard was given ivermectin at 1000 µg/kg orally. Within three days, pentastomid ova were no longer present in the fecal exams. The lizard displayed no signs of toxicity and continued to feed well. The male cage-mate was also given the higher dose of ivermectin with no apparent ill effects. The pair successfully bred and reproduced on two occasions. Fecal examinations on one offspring did not demonstrate pentastomid ova. The female continued to exhibit no pentastomid ova on weekly fecal exams for 10 months and was then sold.

Since this animal was not sacrificed and necropsied, it could not be determined whether the ivermectin eliminated the pentastomids or merely halted ova production. The lizard was given the higher dose of ivermectin as a last resort. The owner did not wish to keep the lizard if it continued to shed pentastomid ova due to this parasite's zoonotic potential. This dose of ivermectin should be used with caution in animals where no previous data are available. Ivermectin toxicities have been reported in other reptile species at much lower dosages.[1]

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Cryptosporidiosis in a Juvenile Freckled Monitor, *Varanus tristis orientalis*

A two-month-old hatchling freckled monitor, *Varanus tristis orientalis*, presented for anorexia and lethargy of one week duration. There was no history of regurgitation or diarrhea.

Physical exam revealed a thin, depressed five-gram monitor. Subjectively, the monitor seemed to have poor bone quality. The mandible seemed to be more pliable when compared to three clinically normal clutch mates.

A fecal exam, fecal culture and antibiotic sensitivities were performed. Fecal flotation and a direct saline smear were negative. Fecal culture demonstrated a few colonies of *Aeromonas hydrophila*, heavy growth of *Escherichia coli* and *Proteus mirabilis*. A fecal acid-fast stain was not performed.

Supportive therapy was initiated with 40 ml/kg prescription diet® canine/feline a/d (Hills Pet Products, Topeka, KS) slurry given by stomach gavage. Note that a/d has a dry matter protein content of 45% and is rich in purines.[1,2] Long term usage might predispose carnivorous reptiles to gout.[1,2] Donoghue[1,2] recommends more suitable diets for sick carnivorous reptiles such as liquid enterals (Ensure®, Ross Laboratories, Columbus, OH) or liquid enterals blended into slurries with either commercial pet foods (such as six oz canned cat food and six oz Ensure®), or prey, such as rodents. Additional supportive therapy consisted of 40 ml/kg Calphosan (Calphosan, 5 mg. calcium glycerophosphate and 5 mg. calcium lactate/ml., Glenwood Inc., Tenably, New Jersey.) intracoelomically. The monitor died that same day.

Gross necropsy revealed subjectively large kidneys with pale foci. No other gross lesions were noted. Tissues submitted for histopathology included brain, stomach, intestines, adrenal gland, kidneys, lung, liver and bone. Histopathology revealed a moderate enteritis/colitis with diffuse cryptosporidiosis. Lesions were not recognized in the stomach. Histologically, the kidneys were normal. The submitted bone was found to have a possible osteopenia. It is not known if the diffuse cryptosporidrosis caused problems with absorption from the intestinal tract and therefore contributed to concurrent emaciation and osteopenia. Diagnostics were not performed on the clinically normal clutch mates due to the owner's wishes.

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